which cannot be controlled by any of the current surgical procedures. Certainly, early recurrent ulceration with pronounced gastric hypersecretion, despite vagotomy and a radical gastric resection, is very suggestive of the Zollinger-Ellison syndrome. Three weeks after discharge, the patient in the present case returned with recurrent ulceration and hypersecretion but a positive Hollander test.

There are other causes of operative failure which imitate ulcerogenic tumors and are far more common. These include incomplete vagotomy and any procedure that interferes with drainage of the antrum.

It must be remembered that recurrent ulcerations may result from ingestion of drugs-for example. cortisone, salicylates or reserpine. More commonly a patient may stimulate gastric secretions by ingestion of alcohol or such caffeine-containing drinks as coffee, tea and cola. Ulcerogenic tumors account for a very small proportion of medical and surgical failures.13

Although incomplete vagotomy and possibly alcoholic intake were thought to be factors in the present case, the patient also returned early with an appearance of recurrent ulcer and hypersecretion. With a pathological diagnosis of islet cell hyperplasia and a clinical diagnosis of Zollinger-Ellison syndrome, total gastrectomy would be the procedure of choice.

Irradiation treatment to the remaining stomach to control secretions has been generally unsuccessful. However, the possibility of avoiding total gastrectomy with a combination of radioactive isotopes (P32), chemotherapy and irradiation has been suggested for certain cases in which there were metastatic ulcerogenic tumors.8

Summary

A case clinically compatible with the Zollinger-Ellison syndrome with pancreatic islet cell hyperplasia is reported. The pertinent literature is reviewed and the surgical management is discussed.

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REFERENCES

- 1. Dreiling, D. A., and Janowitz, H. D.: The effect of glucagon on gastric secretion in man, Gastroenterology, 36:580-581, May, 1959.
- 2. Ellison, E. H.; The ulcerogenic tumor of the pancreas, Surgery, 40:147-170, July 1956.
- 3. Gregory, R. A., Tracy, H. J., French, J. M., and Sircus, W.: Extraction of a gastrin-like substance from a pancreatic tumor in a case of Zollinger-Ellison syndrome, Lancet, 1:1045-1048, May, 1960.
- 4. Poth, E. J., and Fromm, S. H.: The relation of pancreatic secretion to peptic ulcer formation. III. The influence of hyperglycemic-glycogenolytic factor, Gastroenterology, 16:490-494, October, 1950.
- 5. Priest, W. M., and Alexander, M. K.: Islet cell tumor of the pancreas with peptic ulceration, diarrhea, and hypokalemia, Lancet, 2:1145-1147, December 7, 1957.

- 6. Rudolph, L. E., Dammen, G. F., and Moore, T. D.: Intractable peptic ulcer and endocrine adenomas with pituitary amphophilic hyperplasia, Surg., 48:170, 1960.
- 7. Summerskill, W. H.: Malabsorption and jejunal ulceration due to gastric hypersecretion with pancreatic islet cell hyperplasia, Lancet, 1:120-123, January, 1959.
- 8. Wilbur, B. C., Lee, H. R., and Jamplis, R. W.: Ulcerogenic tumors of the pancreas: Report of two cases and suggested treatment, Surgical Clinics No. Am., 43:(5) 1343-1348, October, 1963.
- 9. Zollinger, R. M.: Endocrine adenomas and peptic ulcer, with special reference to pancreatic adenomas, gastroenter-ology, 39:541-543, November, 1960.
- 10. Zollinger, R. M.: Observations on the relationship of the pancreas to peptic ulcer, Bull. of the New York Acad. Med., 39:617-628, October, 1963.
- 11. Zollinger, R. M., and Craig, T. V.: Endocrine tumors and peptic ulcer, Am. J. Med., 29:761-768, November, 1960.
- 12. Zollinger, R. M., and Craig, T. V.: Endocrine tumors and peptic ulcer, Am. J. Surg., 99:424-432, April, 1960.
- 13. Zollinger, R. M., and Elliott, D. W.: Ulcerogenic tumors of the pancreas and management of pancreatitis, Med. Science, 12:857-878, November 25, 1962.
- 14. Zollinger, R. M., Elliott, D. W., Endahl, G. L., Grant, G. N., Goswitz, J. T., and Taft, D. A.: Origin of the ulcerogenic hormone in endocrine induced ulcer, Ann. Surg., 156:570-578, October, 1962.
- 15. Zollinger, R. M., and Ellison, E. H.: Primary peptic ulcerations of the jejunum associated with islet cell tumors of the pancreas, Ann. Surg., 142:709-728, October, 1955.
- 16. Zollinger, R. M., and McPherson, R. C.: Ulcerogenic tumors of the pancreas, Am. J. Surg., 95:359-365, March,

GLAUCOMA Secondary to Local Steroid Therapy

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WITH THE WIDESPREAD USE of local steroid therapy for a number of ocular conditions, complications secondary to the treatment have become evident. In recent years, the increasing incidence of serious complications of herpes simplex of the cornea has been laid to activation or stimulation of the virus by steroids.

Recently it has been reported that glaucoma may be a complication of the local use of steroids in the eye.4 Reports have noted increase in ocular tension

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associated with at least four different steroids.2,3,4 Another steroid was found to cause glaucoma in the cases presented here, and it is probable that this is a potential complication of all these drugs. The incidence of this complication is not yet known.

Reports of Cases

CASE 1. The patient was a white man 52 years of age with complaint of redness of the right eye and sensitivity to light for two days. On examination a low grade aqueous flare was noted in the anterior chamber and there were numerous fine cellular keratitic precipitates on the posterior corneal surface. A diagnosis of iritis was made and atropine drops twice a day and Maxidex® (dexamethasone) drops every two hours were prescribed. The response was prompt and the treatment was gradually decreased in frequency. Atropine was discontinued and as the eye was clear the patient was instructed to decrease the Maxidex dosage to zero over the next five days. He reported by phone that he could not keep an appointment the next week but said he had no further problems. About two months later, pain and irritation of the eye developed. At this time, the cornea and anterior chamber were found to be clear. The tension, however, was 39 mm of mercury in the right eye and 17 mm in the left. Gonioscopy showed a wide angle with no synechiae. On questioning, the patient said that, as the Maxidex "felt good," he had continued using it two or three times a day in the right eye. He had not used atropine. He was told not to use Maxidex. Topical instillation of a 0.5 per cent pilocarpine solution was prescribed, and the tension having decreased to 16 mm six days later, this treatment was discontinued. From then on the tension was in a normal range comparable to that in the other eye.

CASE 2. A 46-year-old male physician was seen for a routine refraction. In the preceding five years he had had repeated episodes of redness in either eve which had been diagnosed as episcleritis. Each time, response to topical steroids had been good.

No abnormalities were noted on ocular examination and tension (Schiotz method) was 13 mm of mercury in each eye.

Nine months later redness developed in the right eye and the patient treated himself with Maxidex® (dexamethasone) drops four times a day. Gradual clearing occurred and after about three weeks he stopped using the steroid. Three days later redness developed in the left eye and he presented himself for examination. Episcleritis was noted in the lower half of the globe of the left eye and the right eye was clear. Tension was 27 mm of mercury in the right eye and 17 mm in the left. A tentative

diagnosis of steroid-induced glaucoma in the right eye was made. The right eye was not treated and zinc-phenylephrine drops were infused in the left. Two weeks later the tension was 12 mm in the right eye and 15 in the left. There was some improvement in the episcleritis. The ocular tension remained normal thereafter.

Discussion

Information is accumulating as to serious potential dangers involved in the local use of corticosteroid drugs in the eye. Editorials in two ophthalmic journals have emphasized this problem.^{1,5} The cases herein reported illustrate the danger of causing glaucoma by steroid administration. Many ophthalmologists have been of the opinion that local steroids should not be prescribed until examination with a slitlamp biomicroscope has been carried out. Now, in addition, patients receiving these drugs locally should have periodic determination of ocular tension.

In both of the cases herein reported, tension promptly returned to normal on cessation of steroid therapy, but in these cases the development of glaucoma was discovered early. Evidence suggests that with increased duration of steroid induced glaucoma, the pressure becomes increasingly elevated, with little response to the usual glaucoma medication. It is not known if the condition will become irreversible with longer duration.

In the belief they are harmless, steroid-antibiotic combinations frequently have been prescribed for any ocular redness or irritation, often without a diagnosis. In view of the possible complications recently reported, they should be employed only on definite therapeutic indication.

Summary

Two cases of unilateral glaucoma occurring with the unilateral use of local steroid therapy are presented. In both instances the condition was noted early and ocular pressure returned to normal on cessation of the steroid.

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REFERENCES

- 1. Allen, H. F.: Quis Custodiet Ipsos Custodes (editorial), Arch. Ophthal., 70:592, 1963.
- 2. Armaly, M. F.: Effect of corticosteroids on intra-ocular pressure and fluid dynamics, Arch. Ophthal., 70: 482, 1963.
- 3. Becker, B. and Mills, D. W.: Corticosteroids and intraocular pressure, Arch. Ophthal., 70:500, 1963.
- 4. Goldmann, H.: Cortisone glaucoma, Arch. Ophthal-
- 5. Thygeson, Phillips: Steroid Therapy in Ophthalmology (editorial), Am. J. Ophthal., 56:668, 1963.